

tation usually indicates disordered physiology and the need for valve surgery, surgery should also be performed if asymptomatic left ventricular dysfunction has begun to develop. The loading conditions in mitral regurgitation are favorable to left ventricular ejection; preload is increased whereas afterload is normal or occasionally decreased, and thus the lesion itself facilitates left ventricular emptying.⁵⁷ Therefore, in the presence of normal muscle function, the ejection fraction should be supernormal in the patient with mitral regurgitation.⁵⁹ Once the ejection fraction falls below 60 percent, the prognosis worsens.⁶⁰

Left ventricular performance can also be gauged in mitral regurgitation by assessing the diameter to which the left ventricle can contract at the end of systole. End-systolic dimension is less dependent on preload than is ejection fraction and can be used as another measure of left ventricular contractile function.⁶¹ When the end-systolic dimension exceeds 45 mm, the prognosis worsens.⁶²⁻⁶⁴ Thus, patients should be referred for surgery if more than mild symptoms develop, or if the ejection fraction falls toward 60 percent or the end-systolic dimension approaches 45 mm, even in the absence of symptoms.

Hochreiter et al.⁶⁵ demonstrated a worsened prognosis if right ventricular function is reduced, emphasizing the prognostic role of pulmonary hypertension in this disease. Patients with a right ventricular ejection fraction of less than 30 percent are at especially high risk.

Importance of the Mitral-Valve Apparatus

Although the importance of the mitral-valve apparatus was described decades ago,⁶⁶ its role in sustaining left ventricular function has become almost universally recognized only recently. Mitral-valve repair has a lower operative mortality and a better late outcome⁶⁷ than mitral-valve replacement. Thus, mitral-valve repair rather than replacement should be performed whenever possible.⁶⁸⁻⁷⁰ Even when the mitral valve must be replaced because of extensive degeneration of the valve, an attempt should be made to conserve the chordal structures and their connections. In the past, when standard replacement of the mitral valve involved destruction of the chordal apparatus, the ejection fraction almost always fell after the operation. Currently, however, the ejection fraction is usually maintained at its preoperative level when the chordal apparatus is preserved in either repair or replacement of the mitral valve.^{69,71,72} Repair rather than replacement also obviates the need for anticoagulant therapy in patients in sinus rhythm and avoids possible failure of the prosthetic valve.

With these improved surgical techniques, postoperative survival after well-timed mitral-valve surgery now approaches that of the general population, as it does for patients with aortic or mitral stenosis.⁶⁰ It

should be noted, however, that whereas in aortic stenosis, age alone is not a contraindication to surgery, in mitral stenosis patients more than 75 years of age have a worse prognosis after surgery than younger patients, especially if mitral-valve replacement rather than repair has been performed or if coronary disease is present.⁷³

Medical Therapy

Although vasodilators are successfully used to increase forward output and decrease left ventricular filling pressure in patients with acute mitral regurgitation, there is currently no apparent benefit to long-term use, especially in asymptomatic patients.⁷⁴ Although such benefit might be possible, no long-term, large studies have demonstrated that the use of vasodilators safely reduces or delays the need for surgery or improves outcome.

AORTIC REGURGITATION

Aortic regurgitation results from disease of either the aortic leaflets or the aortic root that distorts the leaflets to prevent their coaptation. Common causes of leaflet abnormalities that result in aortic regurgitation include infective endocarditis and rheumatic fever. Aortic-root causes of aortic regurgitation include annuloaortic ectasia (idiopathic root dilatation associated with hypertension and aging), Marfan's syndrome, aortic dissection, collagen vascular disease, and syphilis.

In chronic aortic regurgitation, left ventricular enlargement produces a large total stroke volume that is entirely ejected into the aorta. In contrast, in mitral regurgitation the regurgitant volume enters the left atrium. Increased stroke volume increases pulse pressure, causing systolic hypertension, which imposes increased afterload on the left ventricle. Indeed, afterload can be as high in aortic regurgitation as it is in aortic stenosis.^{75,76}

Recognition and Assessment of Severity

The large total stroke volume in aortic regurgitation increases pulse pressure, which produces a myriad of clinical signs. Although the typical diastolic blowing murmur heard along the left sternal border is the usual sign of aortic regurgitation, the peripheral signs of a hyperdynamic circulation often indicate that the disease is severe. A partial list of these signs includes Quincke's pulse (systolic plethora and diastolic blanching in the nail bed when gentle pressure is placed on it), Corrigan's pulse (a bounding, full carotid pulse with a rapid downstroke), Musset's sign (head bobbing), and Hill's sign (systolic blood pressure in the leg at least 30 mm Hg higher than that in the arm).

In addition to the typical murmur of aortic insufficiency, a diastolic rumble (Austin Flint murmur) may also be heard over the cardiac apex. Although

its origin is debatable, the Austin Flint murmur is probably produced as the aortic jet impinges on the mitral valve, causing it to vibrate; also, simultaneous diastolic filling of the left ventricle from the left atrium and aorta tends to close the mitral valve in diastole, producing physiologic stenosis.

Once aortic regurgitation is suspected on physical examination, echocardiography with Doppler examination of the aortic valve can help estimate its severity. Aortography during catheterization helps confirm the severity of the disease.

Therapy

Surgical Correction

As with mitral regurgitation, symptoms may not appear until left ventricular dysfunction in aortic insufficiency is well advanced. The symptoms are usually those of left-sided heart failure (dyspnea, orthopnea, and fatigue). Angina may also occur in patients with aortic insufficiency without coronary disease, but less frequently than in patients with aortic stenosis.¹³ Although aortic insufficiency should be corrected when more than mild symptoms develop, there is compelling evidence that aortic regurgitation should be corrected before the onset of permanent left ventricular damage, even in asymptomatic patients.⁷⁷⁻⁸¹ As noted above, aortic insufficiency increases left ventricular afterload, in part because the high stroke volume produces a wide pulse pressure and systolic hypertension. After aortic-valve replacement, afterload is reduced and ejection fraction improves. Thus, it is not surprising that patients with aortic insufficiency can have a greater decrease in ejection performance and a larger end-systolic dimension than patients with mitral insufficiency, while still having a good postoperative outcome.

In general, the "55 rule" has been useful in gauging the timing of surgery for this disease.⁷⁷⁻⁸¹ Aortic-valve surgery should be performed before the ejection fraction falls below 55 percent or the end-systolic dimension exceeds 55 mm. The markers for the timing of surgery in mitral regurgitation and aortic insufficiency are shown in Table 1. Although replacement of the aortic valve with a tissue or mechanical prosthesis has been the definitive therapy

for severe aortic regurgitation, experience with the pulmonary autograft (Ross procedure) and aortic-valve reconstruction is rapidly increasing.⁸²⁻⁸⁵

Medical Therapy

Because aortic regurgitation represents a state of excess afterload, it could be anticipated that reduction of afterload with vasodilators would improve left ventricular performance while simultaneously decreasing the amount of aortic regurgitation, thus reducing or delaying the need for surgery. The most compelling evidence supporting this concept is from a study showing that the use of nifedipine in asymptomatic patients with severe aortic regurgitation and normal left ventricular function can delay the need for surgery by two to three years.⁸⁶ It is likely that other vasodilators will also be efficacious in safely forestalling surgery.⁸⁷

Acute Aortic Insufficiency

Assessment of Severity and Timing of Surgery

Acute severe aortic insufficiency is usually a surgical emergency. The large regurgitant volume suddenly entering the left ventricle, before adaptation to the volume load has developed, increases left ventricular filling pressure, causing acute pulmonary congestion. Severe regurgitation impairs forward cardiac output, thus reducing organ perfusion. Reduced output, in concert with elevated left ventricular filling pressure, probably reduces coronary blood flow, possibly potentiating myocardial ischemia and further left ventricular deterioration. A fact of diagnostic importance is that the large left ventricular stroke volume present in compensated chronic aortic insufficiency is absent in acute aortic insufficiency, because left ventricular enlargement has not yet occurred. Therefore, many of the signs of severe aortic regurgitation discussed above are absent, and the diagnosis is easy to miss.⁸⁸

Important clues during physical examination include the diastolic blowing murmur of aortic insufficiency and a soft first heart sound. A soft first heart sound occurs because rapid ventricular filling due to aortic insufficiency closes the mitral valve before the onset of systole, and thus S₁ is constituted only by the closure sound of the tricuspid valve. Preclosure of the mitral valve, suspected on physical examination and confirmed by echocardiography, is an ominous development, usually indicating the need for urgent surgery.⁸⁹

Because acute aortic insufficiency is usually caused by infective endocarditis, there is always concern about aortic-valve replacement in the presence of infection. However, in most cases the risk of sudden death from cardiac causes outweighs the relatively small risk (less than 10 percent) of prosthetic-valve infection.⁹⁰ Most consider the aortic homograft the

TABLE 1. ECHOCARDIOGRAPHIC PREDICTORS OF GOOD OUTCOME IN AORTIC AND MITRAL REGURGITATION.

TYPE OF REGURGITATION	END-DIASTOLIC DIMENSION	EJECTION FRACTION	SHORTENING FRACTION
Aortic	≤55	≥0.55	>0.27
Mitral	≤45	≥0.60	>0.32

valve of choice in this situation. Thus, aortic-valve replacement should be contemplated in any patient with acute aortic insufficiency who has evidence of even mild congestive heart failure or mitral-valve preclosure.

CORONARY ARTERY DISEASE

The presence of coronary disease in patients with either mitral or aortic valve disease worsens the long-term prognosis.^{73,91,92} Although the operative risk may not be increased,⁹³ the long-term prognosis in combined coronary and valvular heart disease is not as good as that in valvular disease alone, even when coronary bypass surgery is performed at the time of valve replacement. This is presumably a result of the progressive nature of coronary disease. Ischemic mitral regurgitation carries the worst prognosis: operative mortality is 10 to 20 percent, and long-term survival is substantially lower than with nonischemic mitral regurgitation.^{94,95}

An unresolved issue is the approach to the aortic valve in patients who have mild-to-moderate aortic stenosis and who are undergoing coronary bypass grafting. Although some centers report that patients who undergo later reoperation because of progression to severe aortic stenosis do not have an increased risk of morbidity and mortality,⁹⁶ others suggest a very high mortality rate.⁹⁷ Because progression to severe aortic stenosis may occur rapidly,⁹⁸ it has been suggested that serious consideration should be given to elective valve replacement at the time of the initial bypass operation.⁹⁷

SUMMARY

The prognosis for patients with valvular heart disease has improved substantially over the past 15 years. A better understanding of the proper timing of surgery is one of the key reasons. In general, surgery for stenotic valvular disease can be delayed until symptoms appear. Conversely, in regurgitant valvular heart disease, prognostically important left ventricular dysfunction may develop in the absence of symptoms, and thus valve surgery for some asymptomatic patients is entirely appropriate.

It is likely that in the future there will be progress toward increasing conservation of the patient's native valve. This will be beneficial because even modern prosthetic valves have inherent risks.⁹⁹ Acquired aortic stenosis will often continue to require prosthetic aortic-valve replacement. However, valvular disease will increasingly be treated by procedures that conserve native valves. These include pulmonary autografts for aortic stenosis, balloon commissurotomy for mitral stenosis, mitral-valve repair for mitral regurgitation, and aortic-valve repair for aortic regurgitation. These procedures will make surgery more attractive by eliminating the risks associated with prostheses. Thus, continuing advances in noninva-

sive assessment of the aortic and mitral valves, appropriate timing of referral for surgery, improved surgical techniques for valve replacement and reconstruction, and very recent advances in less invasive surgical approaches should combine to improve the outlook for patients with valvular heart disease.

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